Airway Resistance

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Airway resistance refers to the degree of resistance to air flow through the respiratory tract during inspiration and expiration. The degree of resistance depends on multiple factors, in particular airway **diameter** and whether flow is **laminar** or **turbulent**. Alveolar expansion is also dependent on **surfactant**, so we will consider the physiology and importance of this substance.

In this article, we shall consider how these factors affect flow, and consider some clinical conditions in which airway resistance is affected.

Determinants of Airway Resistance

Certain equations can be used to determine airway resistance.

Ohm's law usually refers to electrical circuits, in which current = voltage/resistance. However, it can also be applied to describe the relationship between airflow, pressure gradient and resistance. The equation is:

$$\text{Flow} = \text{Pressure gradient} / \text{Resistance}$$

This demonstrates that as resistance increases, the pressure gradient must also increase to maintain the same rate of flow into the alveoli.

Diameter

Poiseuille's Law, also known as the Hagen–Poiseuille equation, gives us the relationship between airway resistance and the diameter of the airway (R = resistance, η = substance viscosity, l = length of tube, r = radius of tube):

$$R = (8 \cdot \eta \cdot l) / (\pi \cdot r^4)$$

In the context of the respiratory system, the components of this equation are very hard to measure accurately. This law only applies when there is **laminar flow**.

However, it shows that airway resistance is **inversely proportional** to the radius, to the power of 4. Therefore, a small change in diameter has a huge effect on the resistance of an airway — e.g. halving the radius of an airway would cause a 16-fold increase in resistance.

Therefore, individually, the smaller airways have much **higher resistance** than larger airways such as the trachea. However, the significant downstream branching of the airways means that there are many

smaller airways **in parallel**. This reduces the total resistance to airflow. So, due to the vast number of bronchioles that are present within the lungs running in parallel, the highest total resistance is actually in the trachea and larger bronchi.

Nervous System Control

The **autonomic nervous system** usually determines airway diameter. **Sympathetic** innervation causes relaxation of bronchial smooth muscle via beta-2 receptors, which causes an **increase in airway diameter** to allow more airflow. This is useful in situations such as **exercise**, when sympathetic stimulation triggers airway muscle relaxation to allow more air into the lungs.

Conversely, **parasympathetic** innervation works on **muscarinic (M3)** receptors to increase smooth muscle contraction and **reduce diameter**.

Inspiration vs Expiration

Boyle's Law states that the pressure of a gas is **inversely proportional** to its volume. During **inspiration**, contraction of the diaphragm and external intercostal muscles causes the thoracic cavity to increase in volume. As the volume of the alveoli and bronchioles increases, intrapulmonary pressure is **reduced**. As external pressure now exceeds internal pressure, air **enters** the lungs down the pressure gradient.

With the increased volume, the intrathoracic pressure surrounding the smaller airways is reduced, allowing for **airway expansion**. As the radius of the airways increases, resistance to airflow is **lower** during this inspiratory phase.

Conversely, in **expiration**, the intrathoracic pressure **increases** due to the lower volume of the thoracic cavity. This pressure leads to the narrowing of the smaller airways, so resistance is **higher** during expiration. In healthy lung tissue, the **elastic fibres** of the surrounding alveoli pull on the walls of small airways and hold them **open** — this force is called **radial traction**.

The higher the elastic recoil of the lungs, the **greater** the radial traction will be. Radial traction helps to **prevent airway collapse** in expiration.

In **forced expiration**, the intrathoracic pressure becomes so high that it causes some smaller airways to **collapse**. Collapsed airways have **exponentially high resistance**. This means air which cannot be expelled is trapped in the alveoli — this contributes to the **residual volume**.

Turbulent vs Laminar Flow

Laminar flow refers to the state of flow in which air moves through a tube in **parallel layers**, with no disruption between the layers, and the central layers flowing with the greatest velocity.

Turbulent flow refers to when air is not flowing in parallel layers, and direction, velocity and pressure within the flow of air become **chaotic**. Turbulent airflow requires a larger pressure difference to maintain flow through the airways. This in turn increases turbulence and therefore resistance.

This means that turbulence leads to the need for a much **greater difference in pressure** to move the air. In physiological terms this means there needs to be an increase in the pressure difference between the

outside air and the air within the lungs. To achieve this the intercostal muscles and diaphragm would need to **work harder** to expand and contract the lungs.

Surface Tension and Surfactant

Lungs contain large amounts of **elastic tissue**, so in the absence of water, they would be very stretchy (**compliant**). However, the humidity of the airways means that the alveoli are lined by **fluid**. This results in **surface tension** limiting the compliance of lung tissue.

Surface tension refers to the tendency of fluid to shrink to the **smallest possible volume**. In water, the pull of **hydrogen bonds** between molecules generates surface tension. Fluid lines the alveolar surface, thus **resisting stretching** of the alveoli. The higher the surface tension, the harder it is for the lungs to stretch. However, this effect is overcome by respiratory **surfactant**.

Type II alveolar cells secrete **surfactant**. It has a **hydrophilic** component which lies in the alveolar fluid, and a **hydrophobic** component which associates with the alveolar gas. This causes surfactant molecules to rise to the surface of the fluid (the **gas/fluid interface**). The surfactant molecules **disrupt the hydrogen bonds** between water molecules on the surface. By doing so, it helps to **overcome surface tension** and allows the alveoli to expand.

It is essential to note that surfactant is most effective at **low** alveolar volumes when the surfactant molecules are **closest together**.

At **higher** alveolar volumes, surfactant molecules are further apart and become **less effective** at reducing surface tension. Therefore, there is resistance to further alveolar expansion. This explains why once the lungs are mostly filled, further lung expansion is difficult.

Laplace’s Law

In addition to aiding lung expansion, **surfactant** is key in preventing the alveoli from collapsing into each other. Laplace's law can be simplified to state that:

“The pressure within a cylinder or sphere is proportional to the surface tension divided by the radius.”

Therefore, we can say that the pressure within large bubbles, e.g. alveoli, should be **lower** than that within smaller bubbles, due to the larger radius. As gas moves from areas of high pressure (small alveoli) to low pressure (large alveoli), we can see that it would be possible for large alveoli to **consume** small alveoli.

However, as discussed above, surfactant becomes **less effective** at **larger** diameters. Therefore, surface tension **remains proportional** to the radius of the alveolus as size increases. In turn, this ensures pressures are relatively similar between adjacent alveoli of different sizes, which prevents them from collapsing into each other.

CLINICAL RELEVANCE
Asthma

In an **asthma exacerbation**, the already narrowed airways (due to mucosal inflammation and smooth muscle hypertrophy) are further constricted due to **increased** smooth muscle tone.

This can decrease the diameter of the airways significantly, causing resistance to airflow to become **very high**. This means the patient must work harder to overcome the increased resistance. This can lead to **turbulent flow**, causing the characteristic **wheeze** of an asthma attack.

Beta-receptor agonists such as **salbutamol** can be given to **reverse** the constriction. This acts on **beta-2 receptors** in the airway, acting on Gs G-proteins to open potassium channels. Allowing potassium channels to open hyperpolarises the cell, making contraction of the smooth muscle less likely and effectively causing **bronchodilation**.

CLINICAL RELEVANCE
Emphysema

In emphysema, there is **destruction of elastin fibres** within alveoli. Therefore, there is less elastic recoil holding open the **smaller airways**, and thus reduced radial traction. This means that during expiration, when the intrathoracic pressure is greater, the smaller airways **collapse** very easily, trapping an **increased volume of air**.

In Chronic Obstructive Pulmonary Disease (**COPD**) the airway obstruction is compounded by **chronic bronchitis**. This causes additional narrowing of airway lumens. People with COPD often exhale through **pursed lips** in an effort to maintain a **high intrapulmonary pressure** and prevent premature collapse of the small airways.

Frequently Asked Questions

What is airway resistance and what factors influence it?

Airway resistance is the opposition to airflow through the respiratory tract during breathing, primarily affected by airway diameter and the nature of airflow (laminar or turbulent). A smaller airway diameter significantly increases resistance, while the branching of airways in parallel helps reduce total resistance.

How does the autonomic nervous system regulate airway diameter?

The autonomic nervous system controls airway diameter through sympathetic and parasympathetic innervation. Sympathetic stimulation relaxes bronchial smooth muscle via beta-2 receptors, increasing diameter, while parasympathetic activity constricts smooth muscle through muscarinic receptors, decreasing diameter.

What role does surfactant play in airway resistance?

Surfactant, secreted by type II alveolar cells, reduces surface tension in the alveoli, enhancing lung compliance and preventing collapse. By disrupting hydrogen bonds in the fluid lining the alveoli, surfactant facilitates alveolar expansion and decreases resistance during breathing.

How does the pressure change during inspiration and expiration affect airway resistance?

During inspiration, increased thoracic volume lowers intrapulmonary pressure, reducing resistance and allowing easier airflow. Conversely, during expiration, decreased volume raises intrathoracic pressure, increasing resistance as smaller airways may narrow or collapse.

What are the clinical implications of increased airway resistance in conditions like asthma and emphysema?

In asthma, airway resistance increases due to inflammation and muscle contraction, leading to difficulty in airflow and wheezing. In emphysema, loss of elastic recoil results in airway collapse during expiration, trapping air and exacerbating resistance, particularly in patients with Chronic Obstructive Pulmonary Disease (COPD).

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